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Case Report

Lethal rupture of post-traumatic aneurysm of the vertebral artery case report

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ABSTRACT

Traumatic aneurysms or dissections of the vertebral artery have been reported in patients who have suffered minor craniofacial injuries in traffic accidents.

A case is reported of ruptured traumatic vertebral artery aneurysm due to closed head injury without without penetrating injuries or skull fractures.

The macroscopic and histological findings relevant to the vertebral wall were compatible with post-traumatic aneurysm; the rupture of the wall was assumed to be caused by sepsis and local infiltration of inflammatory cells.

1. Introduction

Vertebral artery aneurysms are rare, representing less than 0.5% of all reported aneurysms; they constitute 0.5–3% of intracranial aneurysms and 20% of posterior circulation aneurysms.[1] They are often located on the intracranial segment of the artery.[2]

In the case of traffic accident victims, patients may suffer traumatic vascular injuries such as traumatic aneurysms or dissections[3] of the vertebro-basilar artery (VAB). Vascular injuries may occur due to blunt cervical trauma particularly by rapid deceleration of the high-speed motor vehicle crashes. Also, several authors have suggested the association of vertebral artery lesions with the anatomy and tortousity of the artery itself.[4]

Histologically, traumatic aneurysms have been classified by Burton according to the types of vascular trauma and aneurysms produced, which are in true aneurysms, false aneurysms, and mixed or dissecting aneurysms.[5–7]

True aneurysms involve three intact arterial wall layers and account for most aneurysms.

One per cent of aneurysms are pseudoaneurysms, which develop as a result of complete or incomplete disruption of the arterial intima, possibly due to trauma-induced necrosis of a section of the arterial wall.

False aneurysms[8] are structurally different from aneurysms; whereas the wall of the aneurysm consists of three layers of tissue, the capsule of false aneurysms consists of adventitia and periadventitial tissues.

We describe a case of ruptured vertebral artery aneurysms due to closed head injuries following a traffic accident, without any penetrating injury or skull fracture.

According to literature, the time interval between trauma and the diagnosis of aneurysm may range from a few hours to as long as 10 years.[9] In the present case, the aneurysm was diagnosed after approximately 45 days.

Rupture of the vertebral artery determined fatal subarachnoid hemorrhage and the death of the patient.

Dissection of the vertebral[4,10] artery is a significant cause of posterior circulation stroke and brain stem ischemia at a young age. Furthermore, subarachnoid hemorrhage secondary to closed head injury is rarely associated with traumatic aneurysms of the posterior circulation.[11]

2. Case report

A 13-year-old female was brought to a local hospital after a motor vehicle accident.

Multiple skin abrasions and serious bilateral femoral and pelvis fractures were detected upon hospital admission. The patient was conscious (GCS = 15).

She underwent surgery with an uneventful recovery.

On the 12th day of hospitalization the patient patient was agitated and tearful and complained of lower limb pain.

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On the 20th day the patient was febrile (max temperature 38°); on day 37, she complained of a persistent headache and nausea and showed signs of diplopia, facial left hemiparesis, and lack of coordination of right upper limb. The brain CT scan showed evidence of hypodense pontine and bulbar lesion, right ventricular dilatation, and occipital horn hemorrhage.

At this time the microbiological examination revealed a pseudomonas infection at the lower limbs, treated with specific therapy. The decision was to follow the patient clinically, and treat conservatively.

On day 46, the patient became comatose (GCS = 3). Angiography showed evidence of a fusiform aneurysm located in the intracranial portion of the right vertebral artery (Fig. 1).

The patient was treated surgically with a ventricular evacuation of the hemorrhage. On the 50th day, tests revealed a systemic infection due to *Pseudomonas aeruginosa*. Further similar intracranial bleeds were followed by the death of the patient, on day 58.

3. Autopsy findings

The patient was a 13-year-old female, weight 55 kg, height 160 cm. No cranial fracture observed at autopsy. The macroscopic examination of the brain showed diffuse edema (brain weight: 1390 g), and signs of the previous craniotomy and a diffused hemorrhage on temporo-parietal cortex.

Subarachnoid hemorrhage was observed on the surface of the occipital cortex, trunk and spinal cervical cord.

The autopsy also showed focal discontinuation of the muscular and tendinous structures between the V and VI cervical vertebrae, without fractures.

Serial cross-sections of the brain revealed dilatation of the ventricular right system, focal intraventricular hemorrhage in the third ventricle, and focal pontine hemorrhage. The examination of the brain vascular system evidenced rupture of a fusiform aneurysm of the right vertebral artery (VA), with a split in the wall of about 0.7 mm from basilar artery (Figs. 2 and 3).

No thromboembolism observed at longitudinal section of VA.

The other brain system circulation was undamaged. Blood in the medullary cavity was detected in the cervical spinal cord.

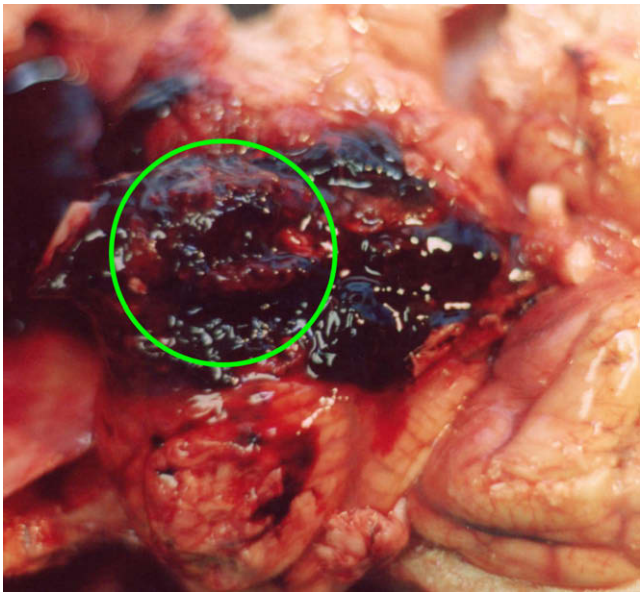

Fig. 1. Posterior view of fusiform vertebral aneurysm showing rupture site.

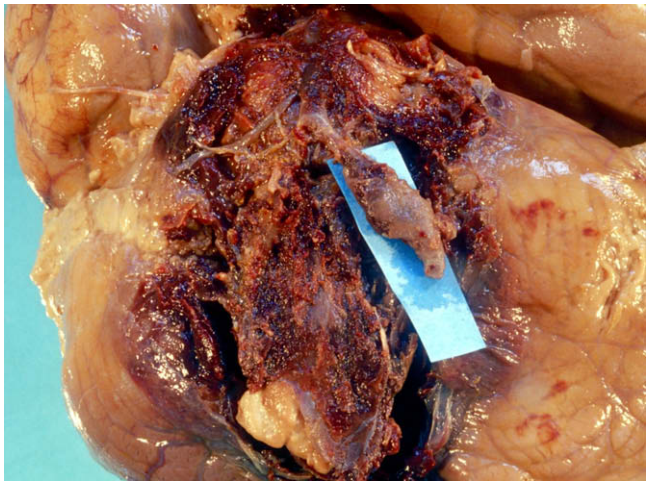

Fig. 2. Fusiform aneurysm of right vertebral artery

The other findings of the autopsy were irrelevant to determine the cause of death.

4. Histological findings

The histological examination showed evidence of inflammatory cell infiltration in the occipital cerebral cortex.

Transmural vasculitis and endoluminal thrombosis were observed in the trunk.

Focal dissection due to diffused subarachnoid hemorrhage and massive inflammatory cell infiltration was detected in the cervical spinal cord.

The right vertebral artery showed evidence of acute suppurative vasculitis and perivasculitis associated to rupture of external elastic lamina, media, and adventitia and segmental disruption of internal elastic lamina (Figs. 4 and 5).

Neutrophil infiltration was observed in the vascular wall around the site of rupture.

5. Discussion and conclusion

Epidemiologically, [12] vertebral artery injuries (VAI) secondary to blunt cervical trauma are rare events.

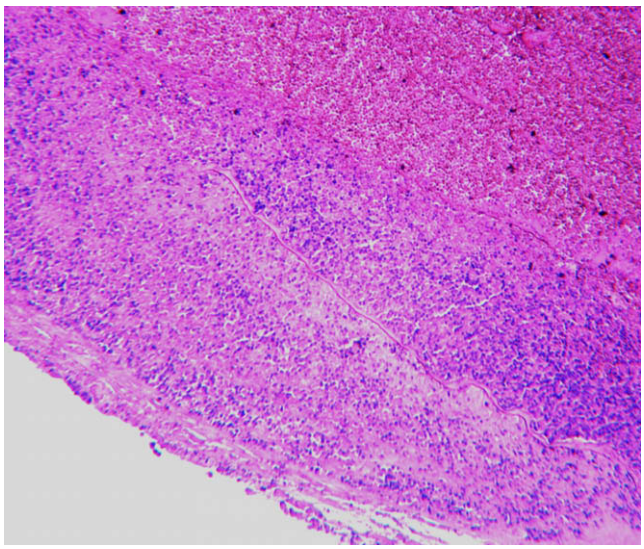

Fig. 3. Photomicrograph of the wall of vertebral artery.

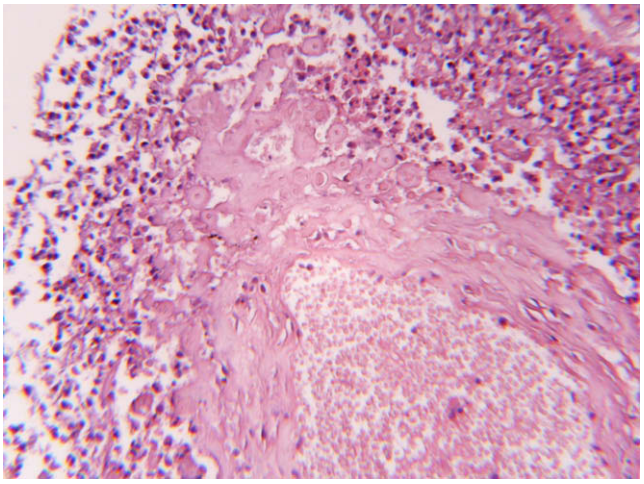

Fig. 4. Photomicrograph of the wall of vertebral artery. Hematoxylin and eosin × 200.

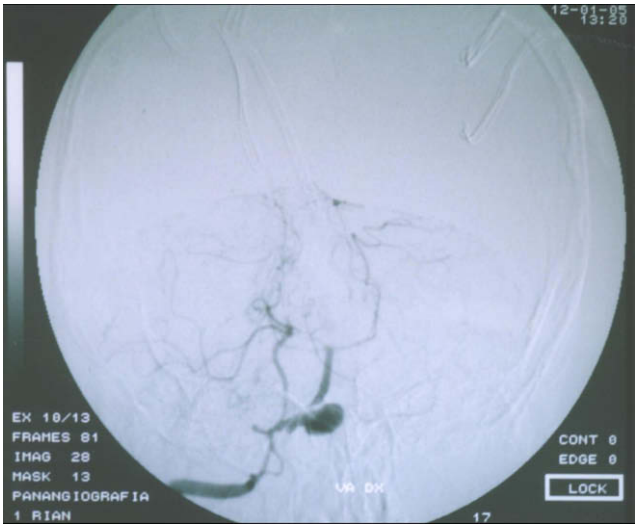

Fig. 5. The angiography showed a fusiform aneurysm located on the right vertebral artery.

Some authors believe that severe blunt cervical trauma[4,11,13] is more likely to cause not only cervical spine injury, but also extensive soft tissue injury and VAI.

A motor vehicle accident (MVA) is the most frequent cause of VAI (*N*50% in most studies), partly because MVA is the first cause of cervical spine injury on its own.

Other authors have associated VAI not only with the duration and force of movement, but particularly with its typology.

In general, a vascular injury after a blunt cervical trauma results either from shearing forces secondary to rotational injuries or from direct trauma to the vessel wall from bony prominences or fragments[4,11,14,15].

Distraction/extension, distraction/flexion, and lateral flexion injuries (classification by Allen et al.) have been implicated as major mechanisms of injury in VAI. Moreover, either distraction/extension or distraction/flexion injuries could easily be elicited by the rapid deceleration of the high-speed motor vehicle crash. The high frequency of MVA in VAI patients might be correlated with the fact that VAI patients were relatively young, the mean age being 30–40 years.

The vertebral artery lesion may be associated with the anatomy and tortousity of the artery itself[4].

Anatomically, the vertebral artery is divided into four arbitrary parts, designated V1 to V4. V1 extends from its origin to its entry into the foramen transversarium of the 6th cervical vertebra (C6). The entire length within the vertebral column is labeled as V2 (extending from the transverse foramen of C6 to C1). V3 extends between its exit from the atlas up to its entry into the foramen magnum and the entire intracranial part is labeled as V4, the intracranial portion of the VA.

The intracranial portion of the VA gives rise to three major branches[9]: the posterior inferior cerebellar artery (PICA), the anterior spinal artery, and the posterior spinal artery.

Most traumatic dissections involve the atlanto-occipital segment, between the C1 and C2 cervical vertebrae (uppermost part of the V2 segment), and between C1 and the entry site into the dura (V3 segment). It is likely that an increased mobility, a poor anchoring into the adjacent tissue, and an increased mechanical torsion and stretch in the C1–C2 region predispose to mechanical injury.[4]

Vertebral artery injuries associated with significant cervical spine injuries, resulting from high-energy trauma, most frequently involved V2, the second segment of the artery that runs through the transverse foramen of C6 to C1. Also, intracranial (V4 segment) VA dissections occur less frequently than extracranial VA dissections.

In the case presented, the cause of death was attributed to the sudden rupture of a post-traumatic vertebral aneurysm with characteristics of a fusiform and intracranial aneurysm. The aneurysm involved the segment between V3 and V4, about one centimeter from the basilar artery.

The clinical course and prognosis[16] of patients with intracranial VA lesions range from a relatively benign course with no neurological complications, to death from severe brain injury.[17]

In this case the rupture of the aneurysm caused a diffused hemorrhage in the posterior cranial and brainstem areas. The patient died due to severe pontine and bulbar injuries, which determined coma and respiratory arrest.

Generally, aneurysms may be congenital or developmental, secondary to infection, atherosclerosis, trauma, or neoplastic invasion.

In this case, it is likely that the neck trauma caused the artery injury in the cervical side, and the subsequent rupture of the aneurysm was favored by a local infiltration of inflammatory cells.

The autoptical examination revealed muscular and tendinous structures discontinuation between the V and VI cervical vertebrae. This lesion is compatible with neck movements such as rotation or flexion-extension as a consequence of the serious traffic accident.

The microscopic examination revealed the disruption of all three layers as in a "true" aneurysm. The histopathology confirmed the local infiltration of inflammatory cells, evidencing the typical thickened adventitia with infiltration of the adventitia and media with clusters of plasma cells and lymphocytes. Giant cells were not identified.

The differential diagnoses for these findings include a non-giant cell variant of giant cell aortitis and nonspecific aortitis.

It is likely that the general infection diagnosed during hospitalization caused the local infection of the aneurysm and favored the alteration and rupture of the arterial wall.

In conclusion, our case seems important for various reasons. First of all, because of the evidence of cervical and cranial trauma in the formation of aneurysms, as in others studies.[18] Secondly, because of the septic infection associated with the local artery infiltration of inflammatory cells, which has caused the rupture of the aneurysm.

Conflict of interest statement

There in no conflict of interest statement.

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Ethical approval

No ethical approval is needed.

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